REVIEWS

1777 Nerve–Cancer Cell Cross-talk: A Novel Promoter of Tumor Progression
Phillip Jobling, Jay Pundavela, Sonia M.R. Oliveira, Séverine Roselli, Marjorie M. Walker, and Hubert Hondermarck

1782 Amino Acid Transporters in Cancer and Their Relevance to “Glutamine Addiction”: Novel Targets for the Design of a New Class of Anticancer Drugs
Yangzom D. Bhutia, Ellappan Babu, Sabarish Ramachandran, and Vadivel Ganapathy

INTEGRATED SYSTEMS AND TECHNOLOGIES

1789 Molecular Portraits of Epithelial, Mesenchymal, and Hybrid States in Lung Adenocarcinoma and Their Relevance to Survival

Precis: An integrative approach combining genomics and proteomics with functional profiling revealed an association between cytoskeletal and actin-binding proteins, a mesenchymal or hybrid EMT phenotype, and invasive properties of lung adenocarcinomas that impact overall survival in patients.

MOLECULAR AND CELLULAR PATHOBIOLOGY

1801 Lung Tumor Suppressor GPRC5A Binds EGFR and Restrains Its Effector Signaling
Shuangshuang Zhong, Huijing Yin, Yueling Liao, Feng Yao, Qi Li, Jie Zhang, Huike Jiao, Yongxi Zhao, Dongliang Xu, Shuli Liu, Hongxing Song, Yong Gao, Jingyi Liu, Lina Ma, Zhi Pang, Ruxiu Yang, Chengyi Ding, Beibei Sun, Xiaofeng Lin, Xiaofeng Ye, Wenzheng Guo, Baohui Han, Binhua P. Zhou, Y. Eugene Chin, and Jiong Deng

Precis: These results reveal how common loss of expression of a tumor suppressive G-protein coupled receptor during lung tumorigenesis promotes malignant development.
**PREVENTION AND EPIDEMIOLOGY**

**1859** miR-21 Inhibition Reduces Liver Fibrosis and Prevents Tumor Development by Inducing Apoptosis of CD24+ Progenitor Cells

Jing Zhang, Jingjing Jiao, Silvia Cermelli, Kyle Muir, Kwang Hwa Jung, Ruhai Zhou, Asif Rashid, Mihai Gagea, Sonya Zabludoff, Raghu Kalluri, and Laura Beretta

Précis: These findings highlight the function of a widely studied oncomiR in the survival of CD24+ tumor-initiating cells and reduced liver fibrosis.

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**1868** Histone Deacetylase Inhibitors Repress Tumoral Expression of the Proinvasive Factor RUNX2

Valentina Sancisi, Greta Gandolfi, Davide Carlo Ambrosetti, and Alessia Ciarrocchi

Précis: These findings offer evidence that the cytotoxic activity of HDAC inhibitors against cancer cells relies not only on reactivating silenced tumor suppressor functions, as widely thought, but also on silencing oncogenes that drive cell survival and malignant progression.

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**1883** RSPO2 Enhances Canonical Wnt Signaling to Confer Stemness-AssOCIated Traits to Susceptible Pancreatic Cancer Cells

Matthias Ilmer, Alejandro Recio Boiles, Ivonne Regel, Kenji Yokoi, Christoph W. Michalski, Ignacio I. Wistuba, Jaime Rodriguez, Eckhard Alt, and Jody Vykoukal

Précis: These results show how blocking a stemness-promoting pathway in conjunction with established chemotherapy could help disrupt dynamic cancer stem-like cell processes and present novel therapeutic targets and strategies.

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**1897** CEACAM1-3S Drives Melanoma Cells into NK Cell-Mediated Cytolysis and Enhances Patient Survival

Nico Ullrich, Anja Heinemann, Elena Nilewski, Inka Scheffrahn, Joachim Klode, André Scherag, Dirk Schadendorf, Bernhard B. Singer, and Iris Helfrich

Précis: These findings define splice isoform-specific immunomodulatory and cell biological functions of cell adhesion protein CEACAM1 in melanoma pathogenesis, shedding light on how different splice isoforms affect the oncogenic versus suppressive actions of this important but complex factor in cancer cells.

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**1908** TWIST1-Induced miR-424 Reversibly Drives Mesenchymal Programming while Inhibiting Tumor Initiation


Précis: These findings identify the first microRNA controlling a plastic tumor cell state that must be regulated up or down at different times during metastatic progression, highlighting the dynamism in epigenetic changes needed for metastasis and hence the inherent complexity needed in therapeutic approaches for advanced cancers.

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**CORRECTION**

**1922** Correction: PTEN Loss Contributes to Erlotinib Resistance in EGFR-Mutant Lung Cancer by Activation of Akt and EGFR

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ABOUT THE COVER

GPRC5A was repressed, while EGFR was dysregulated, in inflammatory lung tissues \( n = 10 \) in comparison with those in normal lung tissues \( n = 10 \). The inverse correlation between EGFR and GPRC5A was complete, without one exception. IHC staining for GPRC5A in human inflammatory lung tissue is shown in the representative image. For details, see article by Zhong and colleagues on page 1801.